# Review of the scientific rationale, development and validation of the International Olympic Committee Relative Energy Deficiency in Sport Clinical Assessment Tool: V.2 (IOC REDs CAT2)—by a subgroup of the IOC consensus on REDs

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## ABSTRACT

Relative Energy Deficiency in Sport (REDs) has various different risk factors, numerous signs and symptoms and is heavily influenced by one's environment. Accordingly, there is no singular validated diagnostic test. This 2023 International Olympic Committee's REDs Clinical Assessment Tool—V.2 (IOC REDs CAT2) implements a three-step process of: (1) initial screening; (2) severity/risk stratification based on any identified REDs signs/symptoms (primary and secondary indicators) and (3) a physician-led final diagnosis and treatment plan developed with the athlete, coach and their entire health and performance team. The CAT2 also introduces a more clinically nuanced four-level traffic-light (green, yellow, orange and red) severity/risk stratification with associated sport participation guidelines. Various REDs primary and secondary indicators have been identified and 'weighted' in terms of scientific support, clinical severity/risk and methodological validity and usability, allowing for objective scoring of athletes based on the presence or absence of each indicator. Early draft versions of the CAT2 were developed with associated athlete-testing, feedback and refinement, followed by REDs expert validation via voting statements (ie, online questionnaire to assess agreement on each indicator). Physician and practitioner validity and usability assessments were also implemented. The aim of the IOC REDs CAT2 is to assist qualified clinical professionals in the early and accurate diagnosis of REDs, with an appropriate clinical severity and risk assessment, in order to protect athlete health and prevent prolonged and irreversible outcomes of REDs.

## INTRODUCTION

Low energy availability (LEA) is the underlying aetiology of a syndrome known as Relative Energy Deficiency in Sport (REDs) and can affect numerous health systems and performance parameters across female and male athletes of all levels and ages.[1 2] Like other multifactorial diseases, such as coronary heart disease,[3] REDs has multiple and diverse risk factors, has numerous signs and symptoms, is influenced by one's environment, is more prevalent in one sex, may present more in certain ethnic groups, and could be genetically influenced.[4]

## WHAT ARE THE FINDINGS?

⇒ The new 2023 International Olympic Committee's Relative Energy Deficiency in Sport Clinical Assessment Tool—V.2 (IOC REDs CAT2) has been developed to enhance the clinical diagnosis, severity/risk stratification and associated training and competition recommendations, for REDs.

⇒ The IOC REDs CAT implements a three-step process, including: (1) initial screening; (2) severity/risk stratification based on any identified REDs signs/symptoms (primary and secondary indicators, appropriately 'weighted' on the evidence) and (3) a physician-led final diagnosis and treatment plan developed with the athlete, coach and their entire health and performance team.

⇒ Iterations of this IOC REDs CAT were developed using athlete testing, feedback and refinement, followed by REDs' expert validation via voting statements, physician and practitioner validity and usability assessments.

## HOW MIGHT IT IMPACT ON CLINICAL PRACTICE IN THE FUTURE?

⇒ The implementation of the IOC REDs CAT2 by an athlete's health and performance team, led by a sports medicine physician with expertise in REDs, should allow for a more accurate and early diagnosis of REDs, with an appropriate clinical severity and risk assessment and associated training and competition recommendations.

⇒ An improved and more timely diagnosis may help to prevent all signs and symptoms of REDs, resulting in enhanced athlete health and performance.

The complexity of REDs necessitates the exclusion of other potential aetiologies in the differential diagnosis for each REDs indicator,[5–7] and to date, there is no singular, validated diagnostic test for REDs. Accordingly, a clinical assessment tool (CAT) can provide a framework to assist clinicians with an evidence-based approach to diagnosing and treating complicated health issues, such as REDs. Since the

| Table 1 | IOC REDs CAT2—key definitions |
|---|---|
| REDs CAT primary indicators | Outcome parameters most consistently resulting from problematic LEA leading to REDs signs and/or symptoms identified in the scientific literature and/or with the greatest measurement validity (ie, sensitivity, specificity) and/or indicative of increased severity and risk of REDs. Accordingly, these indicators hold the most evidence and impact in the overall IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. |
| REDs CAT secondary indicators | Outcome parameters with some scientific evidence, resulting from problematic LEA leading to REDs signs and/or symptoms identified in the scientific literature and/or with lower measurement validity (ie, sensitivity, specificity) and/or have shown less severity and risk of REDs. Accordingly, these indicators hold a secondary level of evidence and impact in the overall IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. |
| REDs CAT potential indicators | Emerging outcome parameters lacking robust scientific evidence but may be linked to problematic LEA leading to REDs signs and/or symptoms. These parameters generally demonstrate many of the following:<br>▶ poor and/or inconsistent evidence<br>▶ no existing validated screening tool, including a lack of validated cut-offs or thresholds in athletes<br>▶ poor measurement validity (ie, sensitivity, specificity, or high variability)<br>▶ high cost and/or poor global availability<br>Accordingly, these indicators are listed as supportive in the severity/risk assessment of REDs but are not directly involved in the IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. Potential indicators may move up to secondary or primary designation or off any list, pending more research validity and/or improved availability and/or cost. |
| REDs symptoms | Any REDs primary, secondary or potential indicator parameter(s) that an athlete directly reports or experiences (eg, pain from a bone stress injury, amenorrhea, depression, hunger, low libido, performance and training plateaus or declines) on the IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. |
| REDs signs | Any REDs primary, secondary or potential indicator parameter that a clinician identifies on the REDs CAT2 Severity/Risk Assessment Tool. A REDs sign may also be a significant individual change in a primary, secondary, or potential indicator from the athlete's baseline within the context of REDs, with or without athlete symptoms (eg, a significant change in sex hormones, resting metabolic rate, cholesterol, etc.) Note: some indicators can be signs and symptoms (eg, amenorrhea). |
| IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool and Sports Participation Guidelines | A clinical tool to assist with identifying the current severity and/or the future risk of REDs that is comprised of an accumulation of primary and secondary indicators of REDs. The Severity/Risk Assessment and Stratification Tool identifies the severity and/or risk of REDs for a given athlete along a spectrum characterised by a traffic-light continuum from healthy (green) to mild (yellow), to moderate (orange), to severe (red) REDs and provides sport participation guidelines for each level. |
| REDs diagnosis | A diagnosis of REDs results from the clinical assessment by a physician with expertise in REDs, utilising information collected from a multidisciplinary team (eg, sports medicine physician, sports dietitian, sports psychologist, sports physiologist), which ideally includes: (1) appropriately validated questionnaires and/or clinical interview; (2) physical assessment and (3) laboratory and imaging data as indicated in the IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. A REDs diagnosis is predicated on excluding other aetiologies in the differential diagnosis for each REDs indicator and ranges from yellow to orange to red severity/risk. |
| CAT, clinical assessment tool; IOC, International Olympic Committee; LEA, low energy availability; REDs, Relative Energy Deficiency in Sport. | |

original 2015 International Olympic Committee (IOC) REDs CAT,[5] scientific advancements in identifying various signs and/or symptoms of REDs have been further developed, thus facilitating the development of this new IOC REDs CAT2.

Although ~200 REDs-related papers have been published since the 2018 consensus,[1] the supporting scientific evidence for some elements within the REDs CAT2 is still emerging. Most of the existing REDs/LEA data have been generated in adult females and males and in study participants who participate in sport from the development to world-class level (tier 2 to 5).[8] There are much less data supporting the use of the REDs CAT2 in individuals pre puberty or during puberty, menopausal/andropausal adults and para athletes. The REDs CAT2 has undergone expert validation via voting statements and REDs expert physician and practitioner validity and usability assessments. Overall, this new IOC REDs CAT2 aims to provide improved usability and validity to allow for earlier detection and diagnosis of REDs. Prevention and early detection with intervention/treatment are central to minimising the risk of more clinically severe outcomes of REDs.[9]

## GENERAL APPROACH TO THE NEW IOC REDS CAT2

This IOC REDs CAT2 features updated definitions associated with various evidence-based levels of REDs signs and symptoms, leading to a diagnosis (table 1) as well as a novel three-step process (figure 1): step 1: initial screening using population-specific REDs questionnaires or clinical interviewing, with individuals deemed at risk going onto; step 2: assessment of various REDs signs/symptoms (primary and secondary indicators; see definitions in table 1) to inform the Severity/Risk Assessment

Tool (table 2) and Stratification with Sport Participation Guidelines (figure 2); data gathered from these steps inform; step 3: a physician-led final clinical diagnosis/stratification and associated treatment plan implementation, ideally integrating a collaborative multidisciplinary REDs health and performance team. To support this assessment, we have provided a supplementary online calculator (online supplemental appendix 1: The IOC REDs CAT2 Severity/Risk Assessment & Stratification Calculator) as well as produced a simplified version of the REDs CAT2 (online supplemental appendix 2: IOC REDs CAT2). The new REDs CAT2 introduces (a) a more clinically nuanced four-level traffic-light (green, yellow, orange and red) severity/risk stratification due to the appreciation that the 2015 CAT[5] yellow light zone had an extensive clinical severity/risk range of very low (a few minor symptoms) to very high (just a few indicators away from full removal from sport participation); (b) certain REDs indicators (table 2) demonstrate *current* REDs severity, while others better predict *future* risk; (c) various REDs indicators may be more helpful if 'weighted' during severity/risk stratification (figure 2) according to level of scientific evidence, validity and usability; and (d) where scientifically supported, some indicators are more useful if thresholds are specified.

### Equity, diversity and inclusion statement

For this IOC REDs CAT2, authors consisted of a diverse group of physicians, physiologists, nutritionists and researchers, featuring three females and three males representing four different countries. The validation steps also engaged eight different sports medicine physicians (from six different countries) and a

**Figure 1** IOC REDs CAT2 three-step protocol including: Step (1) Screening; Step (2) Severity and Risk Assessment and Stratification; and Step (3) Clinical diagnosis and treatment. CAT, clinical assessment tool; IOC, International Olympic Committee; REDs, Relative Energy Deficiency in Sport.

collection of 16 registered sports dietitians, coaches, sports physiologists, strength and conditioning coaches, and mental performance consultants from eight different countries.

## Step (1): initial REDs screening: questionnaire and clinical interview

Initial screening interventions (ie, questionnaires or interviews) often lack sensitivity and/or specificity when assessing complex multifactorial syndromes via self-reported symptoms without contextual biomarkers (tables 1 and 2). Although questionnaires can be inexpensive, anonymous and scalable to large populations, many lack validation, population-specific normative values and can result in survey fatigue as well as conscious or unconscious over-reporting or under-reporting.[10] Furthermore, although LEA is the underlying aetiology of REDs, it is time-consuming and challenging to accurately quantify and does not reflect past (potentially underpinning) behaviour that has led to the current state of health. Thus, these calculations are generally not recommended for REDs severity/risk assessments.[11][12] Accordingly, there is currently no validated questionnaire that would address all potential REDs symptoms in a single assessment.

Expert clinical interviewing focused on assessing some of the physiological and/or psychological dimensions of problematic LEA (for the definition of problematic LEA, see Mountjoy et al[1]) can circumvent some of the challenges of questionnaires—as experts can further clarify and explore answers with further questioning. However, clinical interviewing is not anonymous, potentially confounded by interviewer and interviewee biases,[13] can be a difficult skill to master for a non-REDs expert and is not scalable to large populations.[14][15]

Nevertheless, some consistent self-reported symptoms have been correlated with LEA, leading to REDs and can potentiate the need for further assessment in step 2 and a final physician-led diagnosis in step 3. The foremost symptoms include a history of bone stress injuries (BSI),[16–22] menstrual cycle dysfunction,[23–27] low libido in males,[28–30] eating disorders/disordered eating (ED/DE) behaviours and various depression-related and anxiety-related symptoms.[27][31–33] It is beyond the scope of this paper to review each tool or questionnaire that has attempted to screen for symptoms, indicating a risk of LEA and/or ED/DE, leading to REDs[34–47] (for a review of these questionnaires, see Tortsveit et al[9]). However, the Low Energy Availability in Females Questionnaire (LEAF-Q)[34] and the Low Energy Availability in Males Questionnaire (LEAM-Q)[36] are two recent tools that have undergone elements of validation in subelite to elite female and male athletes (mainly endurance and weight-sensitive sports). Taken together, we recommend selecting an appropriate

tool, questionnaire or combination of questionnaires that is ideally population validated (eg, sex-specific, age-specific, sport-specific), appreciating sensitivity and specificity, and, if possible, followed by a clinical interview. We recommend that a compulsory component of every athlete's annual preparticipation evaluation includes initial screening for REDs (step 1) and education to increase recognition of various LEA/REDs symptoms by athletes, coaches, parents and members of the athlete health and performance team.[9]

## Step (2): REDs severity/risk assessment and stratification with sport participation guidelines

The following subsections explore the scientific evidence supporting each REDs CAT2 primary, secondary and emerging indicators (table 2; table 1). Herein, we report evidence from short-term, medium-term and long-term REDs studies and across various methodological designs: (1) prospective, controlled investigations (expert-controlled LEA exposures to demonstrate causative relationships to LEA outcomes); (2) longitudinal studies (observations involving careful dietary and exercise records of athletes showing potentially causative relationships via LEA indicators) and (3) cross-sectional observations (demonstrating correlations between LEA surrogates and indicators). This review will not focus on methodological best practices (please see: Ackerman et al[11]). Apart from menstrual function (female only) and testosterone (male only), indicators apply to both sexes.

### Primary indicators
#### Primary and secondary amenorrhea (including oligomenorrhea; females)

Although the term Abnormal Uterine Bleeding with Ovulatory dysfunction is now the preferred and established term in the gynaecology field,[48–50] we will continue to use the more standard terms of various ovulatory and menstruation dysfunctions used in the Female Athlete Triad (Triad), endocrinologic and REDs literature.[39][51][52] In the non-gynaecological fields of medicine, functional hypothalamic amenorrhea (FHA) remains one of the strongest, most explored and well-known consequences of problematic LEA.[1] Since menstrual dysfunction is a medium-term (weeks to months) adaptation to LEA, most investigations using amenorrhea as a surrogate of LEA have been observational or cross-sectional.[53][54] However, hormonal adaptations underpinning FHA have been reported after short-term, controlled LEA.[52] For example, healthy, habitually

**Table 2** IOC REDs CAT2 Severity/Risk Assessment Tool that implements primary, secondary and potential indicators into a traffic-light criterion outlined in figure 2

| REDs indicator | References |
|---|---|
| **Severe primary indicators (count as two primary indicators)** | |
| Primary amenorrhea (*Females*: primary amenorrhea is indicated when there has been a failure to menstruate by age 15 in the presence of normal secondary sexual development (two SD above the mean of 13 years), or within 5 years after breast development if that occurs before age 10; or prolonged secondary amenorrhea (absence of 12 or more consecutive menstrual cycles) due to FHA | 5 39 51 163 181 |
| Clinically low free or total testosterone (*Males:* below the reference range) | 53 82 83 182–184 |
| **Primary indicators** | |
| Secondary amenorrhea (*Females*: absence of 3–11 consecutive menstrual cycles) caused by FHA | 5 39 51 163 |
| Subclinically low total or free testosterone (*Males*: within the lowest 25% (quartile) of the reference range) | 53 82 83 182–185 |
| Subclinically or clinically low total or free T3 (within or below the lowest 25% (quartile) of the reference range) | 53 182 186 |
| History of ≥1 high-risk (femoral neck, sacrum, pelvis) *or* ≥2 low-risk BSI (all other BSI locations) within the previous 2 years *or* absence of ≥6 months from training due to BSI in the previous 2 years | 39 115 |
| Pre-menopausal females and males <50 years old: BMD Z-score* <-1 at the lumbar spine, total hip, or femoral neck or decrease in BMD Z-score from prior testing<br>Children/adolescents: BMD Z-score* <−1 at the lumbar spine or TBLH or decrease in BMD Z-score from prior testing (can occur from bone loss or inadequate bone accrual). | 116 117 119 120 |
| A negative deviation of a paediatric or adolescent athlete's previous growth trajectory (height and/or weight) | 174 175 |
| An elevated score for the EDE-Q global (>2.30 in females; >1.68 in males) and/or clinically diagnosed DSM-5-TR-defined Eating Disorder (*only 1 primary indicator for either or both outcomes*) | 122 124 130 133 134 187 |
| **Secondary Indicators** | |
| Oligomenorrhea caused by FHA (>35 days between periods for a maximum of 8 periods/year) | 5 39 51 163 |
| History of 1 low-risk BSI (see high vs low-risk definition above) within the previous 2 years *and* absence of <6 months from training due to BSI in the previous 2 years | 39 115 |
| Elevated total or LDL cholesterol (above reference range) | 81 135 139 |
| Clinically diagnosed depression and/or anxiety (*only 1 secondary indicator for either or both outcomes*) | 33 124 188 |
| **Potential Indicators (not scored, emerging)†** | |
| Subclinically or clinically low IGF-1 (within or below the lowest 25% (quartile) of the reference range) | 182 189 190 |
| Clinically low blood glucose (below the reference range) | 130 189 |
| Clinically low blood insulin (below the reference range) | 23 24 182 |
| Chronically poor or sudden decline in iron studies (eg, ferritin, iron, transferrin) and/or haemoglobin | 17 191–193 |
| Lack of ovulation (via urinary ovulation detection) | 26 51 65 162 |
| Elevated resting AM or 24-hour urine cortisol (above the reference range or significant change for an individual) | 23 24 182 194 |
| Urinary incontinence (*Females*) | 195–197 |
| GI or liver dysfunction/adverse GI symptoms at rest and during exercise | 27 34 198 |
| Reduced or low RMR <30 kcal/kg FFM/d or RMR ratio <0.90 | 153 158 186 199 |
| Reduced or low libido/sex drive (especially in males) and decreased morning erections | 28–30 36 |
| Symptomatic orthostatic hypotension | 171 174 200 |
| Bradycardia (HR <40 in adult athletes; HR <50 in adolescent athletes) | 171 174 175 |
| Low systolic or diastolic BP (<90/60 mm Hg) | 172 176 |
| Sleep disturbances | 75 201 202 |
| Psychological symptoms (eg, increased stress, anxiety, mood changes, body dissatisfaction and/or body dysmorphia) | 27 32 33 122 124 188 |
| Exercise dependence/addiction | 122 130 203 204 |
| Low BMI | 39 174 175 |

Every indicator below requires consideration of a non-LEA-mediated differential diagnosis. All indicators apply to females and males unless indicated. Menstrual cycle status and endogenous sex hormone levels cannot be accurately assessed in athletes who are taking sex hormone-altering medications (eg, hormone-based contraceptives), and thyroid hormone status indicators cannot be accurately assessed in athletes who are taking thyroid medications. All laboratory values should be interpreted in the context of age-and sex-appropriate and laboratory-specific reference ranges. Most REDs data and associated thresholds have been established in pre-menopausal/andropausal adults unless indicated. Disclaimer: *This tool should not be used in isolation nor solely for diagnosis, as every indicator requires clinical consideration of a non-LEA-mediated differential diagnosis. Furthermore, the tool is less reliable in situations where it is impossible to assess all indicators (eg, menstrual cycle status in females who are using hormonal contraception). This tool is not a substitute for professional clinical diagnosis, advice and/or treatment from a physician-led team of REDs health and performance experts*.

*BMD assessed via DXA within ≤6 months. In some situations, using a Z-score from another skeletal site may be warranted [eg, distal 1/3 radius when other sites cannot be measured or including proximal femoral measurements in some older (>15 years) adolescents for whom longitudinal BMD monitoring into adulthood is indicated].[119 121] A true BMD decrease (from prior testing) is ideally assessed in comparison to the individual facilities DXA's Least Significant Change (LSC) based on the facilities calculated coefficient of variation (%CV). As established by ISCD, at the very least, LSC should be 5.3%, 5.0% and 6.9% for the lumbar spine, hip and femoral neck to detect a clinical change.[120 121]

†Potential indicators are purposefully vague in quantification, pending further research to quantify parameters and cut-offs more accurately.

Adolescent, <18 years of age; BMD, bone mineral density; BMI, Body Mass Index; BP, blood pressure; BSI, bone stress injuries; DSM-5-TR, Diagnostic and Statistical Manual of Mental Disorders, 5th edition, text revision; DXA, dual-energy X-ray absorptiometry; EDE-Q, Eating Disorder Examination Questionnaire; FFM, fat-free mass; FHA, functional hypothalamic amenorrhea; GI, gastrointestinal; HR, heart rate; IGF-1, insulin-like growth factor 1; kcal, kilocalories; LDL, low-density lipoprotein; RMR, resting metabolic rate; T3, triiodothyronine; T, testosterone; TBLH, total body less head.
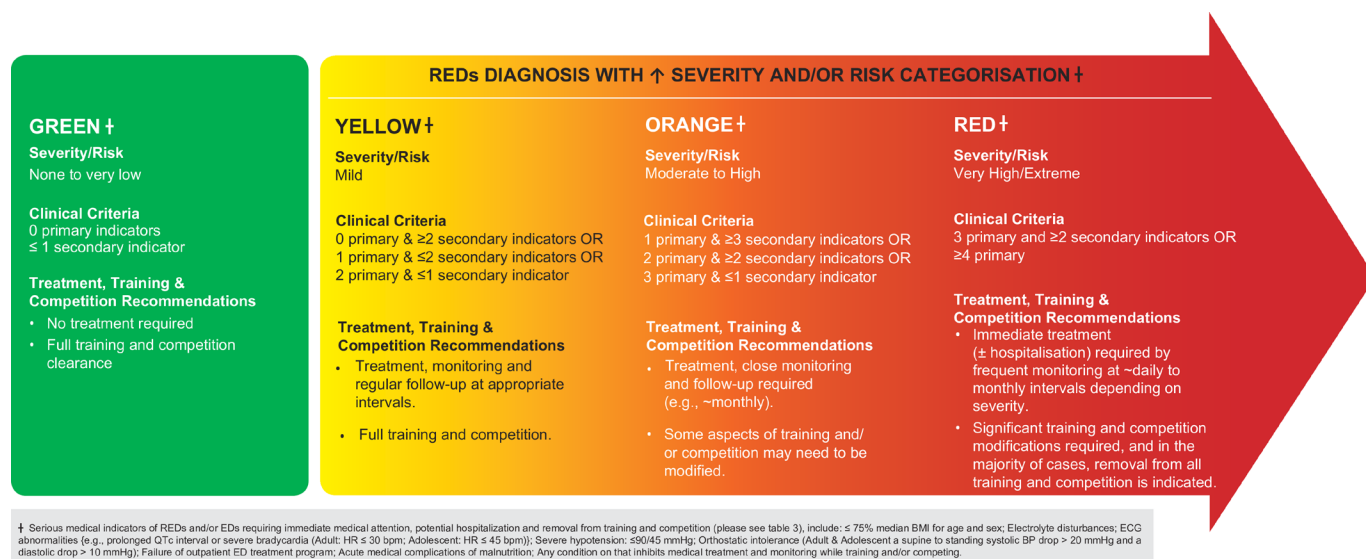
**REDs DIAGNOSIS WITH ↑ SEVERITY AND/OR RISK CATEGORISATION ‡**

**GREEN ‡**

**Severity/Risk**
None to very low

**Clinical Criteria**
0 primary indicators
≤ 1 secondary indicator

**Treatment, Training & Competition Recommendations**
- No treatment required
- Full training and competition clearance

**YELLOW ‡**

**Severity/Risk**
Mild

**Clinical Criteria**
0 primary & ≥2 secondary indicators OR
1 primary & ≤2 secondary indicators OR
2 primary & ≤1 secondary indicator

**Treatment, Training & Competition Recommendations**
- Treatment, monitoring and regular follow-up at appropriate intervals.
- Full training and competition.

**ORANGE ‡**

**Severity/Risk**
Moderate to High

**Clinical Criteria**
1 primary & ≥3 secondary indicators OR
2 primary & ≥2 secondary indicators OR
3 primary & ≤1 secondary indicator

**Treatment, Training & Competition Recommendations**
- Treatment, close monitoring and follow-up required (e.g., ~monthly).
- Some aspects of training and/or competition may need to be modified.

**RED ‡**

**Severity/Risk**
Very High/Extreme

**Clinical Criteria**
3 primary and ≥2 secondary indicators OR
≥4 primary

**Treatment, Training & Competition Recommendations**
- Immediate treatment (± hospitalisation) required by frequent monitoring at ~daily to monthly intervals depending on severity.
- Significant training and competition modifications required, and in the majority of cases, removal from all training and competition is indicated.

‡ Serious medical indicators of REDs and/or EDs requiring immediate medical attention, potential hospitalization and removal from training and competition (please see table 3), include: ≤ 75% median BMI for age and sex; Electrolyte disturbances; ECG abnormalities (e.g., prolonged QTc interval or severe bradycardia (Adult: HR ≤ 30 bpm; Adolescent: HR ≤ 45 bpm)); Severe hypotension: ≤90/45 mmHg; Orthostatic intolerance (Adult & Adolescent a supine to standing systolic BP drop > 20 mmHg and a diastolic drop > 10 mmHg); Failure of outpatient ED treatment program; Acute medical complications of malnutrition; Any condition on that inhibits medical treatment and monitoring while training and/or competing.

**Figure 2** IOC REDs CAT2 Severity/Risk Stratification and Sport Participation Guidelines implementing the associated IOC REDs Severity/Risk Assessment tool (see table 2), with varying clinical management recommendations. Disclaimer: These guidelines are not to be used in isolation and are not to be solely used for diagnosis. Furthermore, these guidelines are less reliable when it is impossible to assess all indicators in table 3. These guidelines are not a substitute for professional clinical diagnosis, advice and/or treatment from a team of REDs health and performance experts led by a physician. Along with the evaluation of health status presented here, severity/risk stratification and sport participation decisions need to be made in the context of various decision modifiers, such as performance level of the athlete, sport type, participation risk, conflict of interest, athlete/ coach pressures, timing and season.[169] BMI, body mass index; BP, blood pressure; CAT, clinical assessment tool; ECG, electrocardiogram; EDs, eating disorders; HR, heart rate; IOC, International Olympic Committee; REDs, Relative Energy Deficiency in Sport.

sedentary females demonstrated altered luteinising hormone (LH) pulse frequency with 4–5 days of energy availability (EA) <30 kcal/kg fat-free mass (FFM)/day[23–25 55]—with similar findings observed after longer weight-loss diets.[56] Seminal research led by Loucks et al found that LEA could cause perturbations in hormone concentrations and menstrual dysfunction, independent of other moderating factors.[23 25 57–59] Longitudinal studies support these findings that implemented LEA leads to an increased prevalence of menstrual dysfunction, both in weight-loss studies of sedentary/untrained females[56 60 61] and in studies of elite physique/fitness athletes losing weight before fitness competitions.[62–64] While no specific EA threshold that has consistently resulted in menstrual dysfunction has emerged, it is clear that decreasing EA increases the likelihood of menstrual dysfunction.[60]

Problematic LEA causes more detrimental outcomes, such as primary FHA and prolonged (>12 months) secondary FHA. Conversely, less severe LEA may result in more subtle forms of menstrual dysfunction (ie, oligomenorrhea or anovulatory cycles). To reflect this continuum, the current CAT2 uses the following scoring system (table 2): primary FHA and prolonged (12 months or longer) secondary FHA=2 primary points; secondary FHA (3–11 consecutive months)=1 primary point; oligomenorrhea (irregular and less frequent cycles)=1 secondary point. We note that the assessment of menstrual cycle status is limited to athletes without any form of hormonal contraception because these can mask menstrual dysfunction,[51 65] and current data do not support the use of hormonal contraception as a treatment strategy to improve bone mineral density (BMD) in premenopausal athletes.[9 66 67] Accordingly, it may be clinically warranted to consider removing an athlete from hormonal contraception to accurately assess menstrual status.

## Testosterone concentrations (males)

Controlled LEA investigations in male athletes are scarce. Those that exist have not shown significant changes to blood testosterone concentrations after short-term (3–5 days at ~15–19 kcal/ FFM/day) LEA.[68 69] However, an 84-hour (3.5 days) military training exercise characterised by very extreme caloric restriction (energy intakes (EI): ~1653 kcal/day with exercise expenditures (EE): ~4000–4500 kcal/day; estimated EA of negative 13 kcal/kg FFM/day) decreased total and free testosterone by 24% and 30%, respectively.[70]

In the medium-term, severe calorie restriction significantly reduces testosterone concentrations across 1–15 weeks of LEA.[71–78] For example, a military investigation showed a 49% and 60% reduction in total and free testosterone concentrations, respectively, after 8 days of high energy expenditures (~3944 kcal/day) combined with restricted EI (~1550 kcal/day; estimated EA of negative 6 kcal/kg FFM/day).[72] Similarly, young males who participated in an 8-week-long military training exercise were reported to have an estimated daily energy deficit of ~1000 kcal, which resulted in an EA of ~14 kcal/kg FFM/day, an ~12% reduction in body mass (BM), and an 86% decline in testosterone concentrations that the authors referred to as 'approaching castrate levels'.[78] Another population habitually and voluntarily exposed to severe LEA are physique and combat sport athletes. Investigations spanning 2–8 months of gradual weight reduction via LEA have repeatedly shown testosterone concentration decreases of 11%–90% (sometimes reaching clinically low levels) in male athletes.[74–77 79 80]

Several recent cross-sectional investigations have chosen 'subclinically low testosterone' as an REDs surrogate for male athletes.[38 53 81] This rationale comes from earlier reviews[82–84] and has proven feasible in subsequent observational research with regards to testosterone and other hormone concentrations (especially thyroid

hormones, please see the next section). Accordingly, we have set cut-offs for REDs hormone concentration indicators to 'clinically low' (below laboratory reference range) and 'subclinically low' (within the lowest 25% ('quartile') of the reference range). While current research supports the use of these specific thresholds for 'subclinically low',[53 81] this is a relatively new concept and we encourage future investigations to refine the 'grey areas' for each specific marker in athlete-specific cohorts. For more information on REDs in males, we refer the reader to the editorial by Hackney[85] within this BJSM REDs special edition.

### Triiodothyronine—T3

A large collection of investigations involving medium to long-term LEA provides strong evidence to support the use of total or free triiodothyronine (T3) as an indicator of LEA.[53 54 62 63 75 86–89] However, there are mixed outcomes with short-term studies, with some in habitually sedentary females reporting total and free T3 reductions after 3–5 days of LEA (8–25 kcal/kg FFM/day)[55 90] while other LEA (~15 kcal/kg FFM/day over 3–5 days) intervention studies in active females[91] and males[68] reporting no change.

Over medium durations, a recent 14-day controlled investigation in male athletes reported reduced T3 and testosterone with extreme LEA (9 kcal/kg FFM/day) but no significant T3 or testosterone reductions with LEA above this threshold.[86] This suggests that longer and/or more severe LEA may be required to induce significant thyroidal and reproductive hormone decrements in male athletes. In the 8-week military training exercise with the ~14 kcal/kg FFM/d EA, mean T3 concentration dropped by 22%.[78] Longitudinal reports in male and female athletes attempting to lose weight via LEA have shown reductions in free or total T3 concentrations in the range of 30% to 67%.[63 75 79 80] These adaptations have not always been reversible after 2 weeks to 5 months of refeeding.[62 63 75]

Long-term adaptations (mainly inferred from cross-sectional studies) involving surrogate markers of prolonged LEA (FHA in female athletes[53 54 87 88 92–96] and subclinically low total testosterone concentrations in male athletes[53]) also have reported reduced free or total T3 compared with healthy counterparts. Among 44 Norwegian male Olympic athletes, 18 had at least one surrogate marker of REDs, and two athletes had subclinically low free T3 accompanied by low resting metabolic rate (RMR), low BMD and subclinically low total testosterone.[81] The cross-sectional assessments of within-day energy balance in female athletes suggest that independent of EA, larger and/or more frequent within-day energy deficits and calorie backloading (50% or more of daily calories consumed after 17:00) are associated with lower total T3 concentrations.[89 97] While most research has shown milder T3 reductions (ie, subclinically low), more severe and/or longer duration LEA (eg, in the face of clinically diagnosed anorexia nervosa) is likely to result in clinically low T3.[98 99]

### BSIs and BMD

Adverse changes to skeletal health (reduced BMD and BSIs) are some of the most insidious outcomes of problematic LEA because they directly impact athlete availability for training and competition, which is directly linked to the likelihood of achieving performance goals.[100] Often years of underlying problematic LEA resulting in low BMD go unnoticed until a BSI occurs. Furthermore, many athletes have surpassed the age of peak bone mass accrual, limiting the ability to achieve 'catch-up' bone mineralisation with EA improvement. Indeed, up to 36% of adult total bone mineral content is acquired during the 4 years surrounding peak height velocity (pubertal growth

spurt,[101]) with peak femoral neck, total hip and lumbar spine BMD attained by age 19–20 years in females and 20–24 years in males.[102] Accordingly, early detection of REDs is critical. There are controlled investigations on the effects of LEA on bone remodelling blood biomarkers,[91 103 104] but it is unclear if these changes reflect longer term adaptations. Therefore, due to lack of knowledge with regards to the time required for bone tissue to experience meaningful changes (ie, altered biomarker concentrations to translate into structural changes), we have only focused on cross-sectional evidence in this section.

It is well established that athletes with apparent REDs (via FHA, male reproductive dysfunction, high-risk LEA on Triad or REDs scoring tool or low body mass index (BMI)) have a higher risk of BSI (1.7 to 4.5-fold increase), low BMD, bone microarchitecture decrements and decreased estimates of bone strength.[16 18–22 27 35 38 53 96 105–115] In a study of adolescent and young adult female athletes, high-risk BSIs were associated with both questionnaire surrogates of LEA and a BMD Z-score < −1.0[116 117] and athletes with all three Triad risk factors (LEA, menstrual dysfunction and low BMD) were 2.5 times more likely to have a high-risk BSI than those with zero risk factors.[115] This is consistent with the increased prevalence of low BMD with higher Triad risk scores in female[17] and male (via modified Triad scoring)[16] athletes. Interestingly, in the latter study, each 1-point increase in cumulative risk score increased the prospective risk of BSI by 37%.

Robust evidence in athletes linking problematic LEA, decreased BMD and increased risk of BSI in athletes make bone health outcomes important components of both primary and secondary indicators. Accordingly, we have implemented the International Society for Clinical Densitometry guidelines and existing literature to identify adult and paediatric low-risk versus high-risk BSI sites and BMD Z-score cut-offs,[39 115 118–121] while also qualifying a previous 2-year window for reporting of BSI's (to reflect *current* REDs severity/risk and minimise athlete recall issues). We have also added a severity criterion; if a BSI causes >6 months absence from sport-specific training over the last 2 years, it is more severe (and a primary indicator; table 2).

### Clinically diagnosed eating disorders or risk of eating disorders

While athletes often experience problematic LEA inadvertently,[122] DE/ED may be an underlying component of REDs. DE/ED is characterised by abnormal thoughts and behaviours around food such as extreme dietary restriction (anorexia nervosa) and/or bingeing and purging (bulimia nervosa). These behaviours highlight the need for early detection of distorted attitudes, beliefs and/or behaviours associated with DE/ED.[122 123] Since DE/ED occurs on a spectrum from disordered behaviours (orthorexia nervosa) to clinically confirmed ED (Diagnostic and Statistical Manual of Mental Disorders, 5th edition, diagnostic criteria text revision,[124]) the assessment of DE/ED in the context of REDs screening and diagnosis incorporates two separate tools: an easily accessible and scored questionnaire, followed by a clinical interview in those identified as at risk for DE/ED.

In female and male athletes, varying forms of dietary and cognitive restraint and body image/shape dissatisfaction (eg, drive for thinness) have been linked to physiological signs of LEA.[22 125–130] For initial screening, we highlight the Eating Disorder Examination Questionnaire (EDE-Q) to identify those at risk for ED in the general population[122 131–133] (questionnaire non-athlete cohort sensitivity (83%) and specificity (96%)).[134] A detailed discussion of DE/ED diagnosis is beyond the scope of the current review.[122] Future research is required to confirm the

validity of EDE-Q and its cut-offs in a wider athletic population (eg, male athletes, transgender and gender nonbinary athletes, and lower risk sports) to improve our understanding of and ability to assess DE/ED in athletes across all sports and genders.

## Secondary indicators

The following section outlines the rationale for inclusion within the CAT2 of the secondary indicators (table 1). Discussion of oligomenorrhea, BMD and BSI has been omitted, given their mention under primary indicators.

### Lipids/cholesterol

Research in females with amenorrhea has associated endothelial dysfunction and unfavourable lipid profiles as a risk for atherosclerosis and elevated low-density lipoprotein (LDL) cholesterol.[135–137] Male case studies observing ~8 weeks of weight loss (via LEA) in preparation for combat sport competition reported elevated cholesterol and LDL levels at the time of competition.[76 77] In addition, an 8-week army course characterised by a significant caloric deficit increased cholesterol levels in male soldiers.[138] In the most extreme cases of starvation (ie, anorexia nervosa), elevated lipid concentrations have been consistently reported.[139] However, evidence from cross-sectional investigations in athletes using various REDs surrogate markers to determine the potential effects of LEA on blood lipids remains inconclusive.[81 94 140]

### Mental health

Anxiety, depression and ED have a high comorbidity rate, but directional effects and casual relationships remain unclear.[141] Among athletes, only a few studies have conducted diagnostic interviews to assess mental disorder prevalence and indicators of ED or REDs.[35 142 143] Accordingly, 28% of female Swedish athletes[143] were clinically diagnosed with an ED concurrently with anxiety and/or affective disorder (ie, depression diagnosed using the Mini-International Neuropsychiatric Interview).[144] In competitive athletes, disordered eating pathology predicted elevated depressive symptoms 6 months later.[145] As such, we recommend assessing the mental health status of athletes, including depressive and anxiety symptoms, when screening for REDs using appropriate validated tools to determine a diagnosis.[144 146–148]

## Potential indicators

This group is characterised as emerging outcome parameters that lack robust scientific evidence but may be linked to LEA, leading to REDs signs/symptoms. Accordingly, these indicators are listed as supportive in the severity/risk assessment of REDs but are not directly involved in the REDs CAT2 Severity/Risk Stratification Tool (table 2). As such, apart from RMR, physique outcomes, ovulation detection, bradycardia, blood pressure (BP) and libido, discussion of all potential indicators in detail are beyond the scope of this paper.

Humans have evolved to adeptly conserve energy during periods of low energy procurement (ie, low EI), or excessive energy expenditure, within a constrained energetic model.[149 150] However, whether increased physical exercise alone, without LEA, is additive or constrained total daily energy expenditure remains debated.[151] Accordingly, it is well established that problematic LEA can result in energy conservation reflected in reduced RMR.[152] While some studies have reported lower RMR in athletes with apparent LEA compared with healthy counterparts,[62 153 154] others have failed to demonstrate this.[81 130 155 156] Challenges relating to the precise measurement of RMR include poor global availability of the method, high variability with poor

measurement validity and reliability, lack of athlete-specific and sport-specific prediction equations and lack of consensus on which thresholds of RMR (absolute or ratio) constitute increased risk.[157 158] While RMR is currently not a component of the REDs CAT2, it is important to note that T3 concentrations have been linked to RMR.[88] Therefore, metabolic adaptation (in the form of reduced T3) is indirectly incorporated into the CAT2.

Another outcome of a constrained energetic model[149 150] is that energetic deficits are not linearly represented in weight loss[150]—with many studies demonstrating a negative exponential relationship/plateau of BM despite continued energetic deficits.[159] Accordingly, while some body composition metrics (eg, large decrease in or extremely low BM, BMI, per cent body fat) can be linked to markers of LEA and REDs (especially over short-term LEA), there are no validated cut-offs established for any body composition metric in relation to REDs severity/risk outcomes when considering sex, chronological and gynaecological age and, most importantly, global ethnicities. Furthermore, the utility of BMI as an accurate medical/health diagnostic has been recently called into question due to a lack of specificity and a failure to account for racial and sex differences[160] or the extreme physiques (with or without health consequences) demonstrated in modern elite sport.[161] Therefore, until further population-specific validation studies are published, body composition metrics remain emerging as a REDs diagnostic indicator.

Exercising females with long-term oestrogen deficiency display impaired regional blood flow and lower systolic BP and heart rate (HR) compared with exercising and sedentary ovulatory females.[137] In a cross-sectional survey of 1000 female athletes, those who screened positively for LEA were over 2.5 times more likely to report suffering cardiovascular issues than non-LEA athletes.[27] Research in males with anorexia nervosa has reported bradycardia and acute systolic heart failure,[98] and a case report of a male bodybuilder and combat sport athlete preparing for competition via implementation of LEA showed reduced brachial BP (132/69 mm Hg to 104/56 mm Hg)[79] and a reduction in resting HR (from ~63 bpm to 35 bpm) across the training period.[154] Although extreme bradycardia and low BP require immediate medical attention (table 3), more data are required to show the utility of these indicators as being sensitive

**Table 3** Serious medical indicators of REDs and/or EDs requiring immediate medical attention, potential hospitalisation and removal from training and competition (adapted from ED clinical management recommendations, paediatric and adult ED papers and athlete cardiovascular health consensus papers[170-176]).

| Serious medical indicators |
| --- |
| ≤75% median BMI for age and sex |
| Electrolyte disturbances (eg, hypokalemia, hyponatremia, hypophosphatemia) |
| ECG abnormalities(eg, prolonged QTc interval or severe bradycardia (Adult: HR ≤30 bpm; Adolescent: HR ≤45 bpm)) |
| Severe hypotension: ≤90/45 mmHg |
| Orthostatic intolerance (Adult & Adolescent: a supine to standing systolic BP drop >20 mmHg and a diastolic drop >10 mmHg) |
| Failure of outpatient ED treatment programme |
| Acute medical complications of malnutrition (eg, syncope, seizures, cardiac failure, pancreatitis) |
| Any condition that inhibits medical treatment and monitoring while training and/or competing |
| *Disclaimer: This list should not be used in isolation and should be based on a thorough clinical assessment that considers the severity of the athlete's physical and mental health.* |
| BMI, body mass index; bpm, beats per minute; ECG, electrocardiogram; ED, eating disorder; HR, heart rate; QTc, corrected QT. |

for the detection of REDS across more mild cases of LEA/REDs in athletes.

An emerging female-specific indicator of REDs is the assessment of ovulation through the associated urinary LH surge via commercial ovulation kits[51][162] (for protocol, see O'Donnell et al[65]). It is important to note that menstrual bleeding does not always indicate ovulation and normal menstrual function. In a study of recreationally active females (running only ~30 km/week), who reported regular menstruation, 12% were found to have anovulatory cycles.[26] However, more research is required to better understand anovulation rates in females who regularly menstruate but have undertaken elite training volumes (20–30 hours/week) and to implement gold-standard methods to elucidate other menstrual cycle dysfunction,[11] while also ascertaining the practicalities of ovulation monitoring in a clinical setting.

Finally, emerging research has shown that decreased libido or attenuation in morning erections may reflect states of LEA in male athletes.[28–30][36] While these assessments would be easy to self-report, we currently lack a validated tool to assess these outcomes and determine what is normal versus indicative of risk.[11]

Step (3): REDs clinical diagnosis and treatment
Accurate diagnosis and an evidence-informed approach to REDs treatment by a multidisciplinary athlete health and performance team, as well as support from the athlete's coach and parents/guardians, are vital to ensure early intervention and to avoid further harmful consequences of problematic LEA.[1][122][163] Ideally, the team should feature a sports medicine physician (if a sports medicine physician is not available, a family physician / general practitioner is preferred), dietitian and mental health consultant, all experienced in REDs diagnosis and management. *With athlete consent*, further support may include the coach, and parents/guardians if the athlete is under 18 years of age, exercise physiologist, therapists, teammates and/or family members. In particular, the athlete's coach should be supportive and aware of the potential impact on training and competition of the treatment plan.[164]

The Severity/Risk Assessment Tool and Stratification with Sport Participation Guidelines (tables 1–3 and figure 2) have been developed to assess the accumulation of short to multiple long-term indicators across several body system domains, leading

to a more accurate diagnosis. As there is no one specific marker to confirm the diagnosis of REDs, there are several other diagnoses that must be excluded, including over-training syndrome,[7] mental health conditions[165] and any endocrine or metabolic disorders not resulting from LEA (eg, autoimmune thyroiditis,[166] malabsorption,[167] primary ovarian insufficiency, polycystic ovary syndrome).[52][168] For further information on various clinical presentations and differential diagnoses, please see table 3 in Torstveit et al[9] and figure 2 in De Souza et al[6] and for further information on REDs methods and testing details (and guidance on lab assays and reference ranges), see Ackerman et al.[11]

The collective results from step 1 to step 2, informed by the Severity/Risk Stratification (table 2) with the 4-colour traffic-light Sport Participation Guidelines (figure 2), are not to be used in isolation but instead require an expert physician-led final diagnosis. We purposefully have a low clinical barrier for diagnosing REDs with a yellow severity/risk traffic-light assessment. Thus, there could be a greater false-positive rate of yellow traffic-light assessments caused exclusively by LEA. But, given the importance of early detection of REDs, type I errors at the lower severity/risk spectrum are warranted. Accordingly, with the accumulation of more indicators required for orange and red traffic-light assessments, the accuracy of a true diagnosis of more severe cases of REDs is improved (reflected in our REDs expert physician validation; figure 3). Along with the evaluation of health status presented here, severity/risk stratification and sport participation decisions also need to be made in the context of various decision modifiers, such as competitive level of the athlete, sport, health risk of continued participation (based on indicators of severity), conflict of interest among those involved in this decision, and intrinsic/extrinsic athlete pressures related to timing in the competition season, desire to compete, sponsorship, athlete's importance to the team and others.[169] Athletes given a red light by the REDS CAT2 are those with evidence of severe endocrinologic or metabolic health disturbances, signs of cardiac involvement, multiple concurrent injuries consistent with RED, and/or behaviours consistent with a severe ED. Depending on the clinical presentation, many of these athletes (and some orange classified athletes) may need training and/or competition adapted or restricted until they have met certain milestones, based on their clinical presentation, indicating no imminent, serious risk to their health.[1][122] Regardless
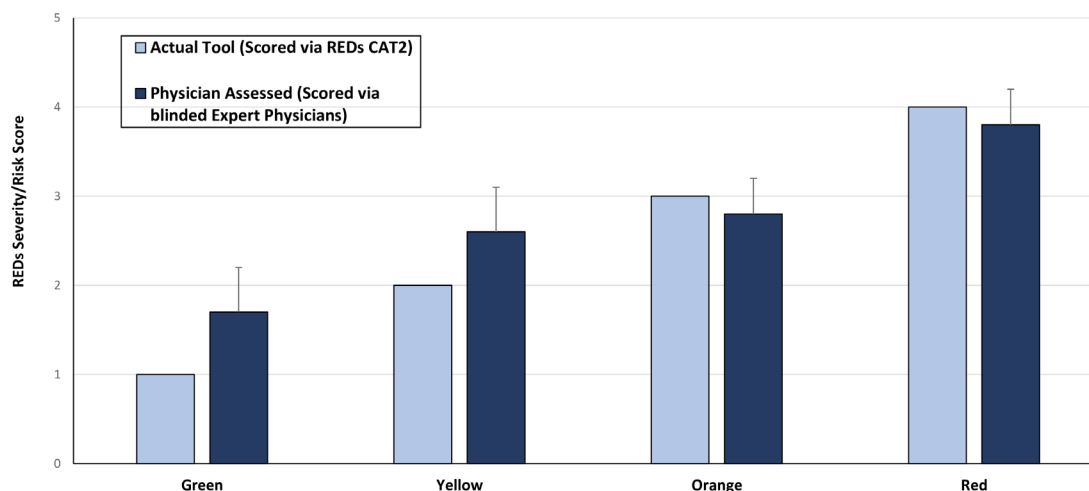


**Figure 3** IOC REDs CAT2 Severity/Risk Assessment Tool Validity. Five different REDs medical and performance-based case studies were assessed via nine blinded (did not receive or use the new IOC REDs CAT2) REDs expert physicians and then compared with actual scoring using the new REDs CAT2 Assessment and Stratification Tools (table 2 and figure 2). The five case studies ranged from green (scored as 1 for correlation and plotting purposes), yellow (scored as 2), orange (scored as 3) to red (scored as 4) in clinical severity/risk (n=40 comparisons of actual vs assessed, r=0.885, p<0.01). CAT, clinical assessment tool; IOC, International Olympic Committee; REDs, Relative Energy Deficiency in Sport.

of their traffic-light assessment, athletes presenting with any serious medical indicators of REDs or EDs require immediate medical attention, potential hospitalisation and removal from training and competition (table 3; serious indicators adapted from ED clinical management recommendations, paediatric and adult ED position papers and athlete cardiovascular health consensus papers).[170–176]

The cornerstone of treatment for all REDs-affected athletes is restoring EA by increasing EI and/or decreasing EEE[9 82 177]; with REDs treatment reviewed in more detail by Torstveit *et al*.[9] This will require an agreed-upon treatment plan, often implementing benchmark goal setting with the athlete, coach and health and performance team members. Consistent messaging and support for the athlete is vital as they navigate changes to their diet and/or training regimen. Athletes with REDs should have clinical follow-ups by the treatment team depending on severity (every 1–3 months, but could be daily or weekly with severe cases featuring imminent risk to athlete health and well-being; figure 2). We recommend athletes with an REDs diagnosis be considered for full re-evaluation with this IOC REDs CAT2 (table 2; figure 2) every ~6 to 12 months (or as appropriate) to confirm diagnostic progress (improvement in traffic-light zone) and restoration of normal body system function. However, certain sections of the tool might be repeated more frequently (eg, blood work) or less frequently (eg, BMD measurement) as clinically indicated.

## IOC REDS CAT2 DEVELOPMENT AND VALIDATION

The development of the IOC REDS CAT2 followed a process from its inception to validation. We developed early draft versions of the CAT2 based on the existing literature, followed by athlete-testing, feedback, refinement and a final REDs expert validation via blinding voting statements (all information on the voting statement process, results, level of agreement and dissenting opinions are presented in the 2023 IOC REDs Consensus[1]), as well as physician and practitioner validity and usability assessments, respectively.

Initially, authors IAH, TS, WTPM, MM and KEA developed early drafts of the presented CAT2 based on purposely weighted diagnostic indicators with the strongest scientific evidence. Early drafts of the CAT2 were then tested in 150+ elite female and male athletes to further refine a draft CAT2 to present at the IOC in-person REDs consensus meeting. Then, utilising the RAND-UCLA Appropriateness Method,[178] a referenced summary of the existing scientific literature was compiled. From this summary, and based on the draft CAT2, evidence-based 'voting statements' on each REDS indicator were developed. The literature summaries and statements were circulated among the REDs expert panel for online confidential round 1 voting following the Delphi method,[179] looking for the level of 'agreement' with the various elements. Panel members were invited to input their rationale for (dis)agreement and/or other ideas.

Third, expert panel members debriefed the voting statement outcomes (from round 1 voting) in person at the Olympic House (Lausanne, Switzerland, September 2022). Statements with <80% agreement, new statements that arose from the discussion and statements that required further clarification underwent a second round of voting. The remaining statements that achieved ≥80% agreement were then incorporated into the final version of the IOC REDs CAT2.

The final step involved the validation of the IOC REDs CAT2 with clinical end users. We first analysed how well the CAT2 aligns with the current expert opinion to overcome this issue. We prepared five athlete case studies, complete with medical history, physical and laboratory data. Eight independent international physicians with expertise in REDs and not involved in the consensus process rated the case studies into low (green), mild (yellow), moderate to severe (orange) and very severe (red) traffic-light zones, *without* receiving the REDs CAT2 scoring tool (table 2) or any further explanations (all case-studies and raw data available on request). Subsequently, we assessed all case studies based on the CAT2 tool and compared our outcomes to the expert opinion. For the physicians, we had 40 assessments (ie, five case studies × eight physicians; see online supplemental appendix 3 for case studies and physician assessment outcomes) and had a very positive, strong and significant correlation between the physician assessment compared with the REDs CAT2 traffic-light outcomes (r=0.885, p<0.01; complete agreement 62±19%; figure 3). Finally, for usability, we asked a range of end users (eg, strength and conditioning coaches, therapists, physicians, psychologists and physiologists) to score the case studies *using* the new IOC REDs CAT2. In this instance, we had 80 assessments (ie, 5 case studies × 16 end users), demonstrating a very strong and positive significant correlation (r=0.963, p<0.01; complete agreement 90±10%). Given the robust scientific rationale for each REDs indicator, voting statements, and various physician validity and practitioner usability assessments, this IOC REDs CAT2 has undergone extensive validation, allowing for a more accurate and precise assessment of the severity/risk of REDs.

## LIMITATIONS

The current CAT2 has made significant diagnostic advances since the 2015 REDs CAT,[5] but is not without limitations. As further highlighted in the IOC REDs companion methods paper,[11] the REDs field suffers from a lack of high-quality placebo-controlled LEA intervention studies. Thus, many conclusions and recommendations are dependent on cross-sectional cohort studies examining markers of LEA and REDs. In some instances, due to lack of athlete cohort data, military and/or anorexia nervosa research has been used to guide selection of indicators. Furthermore, most REDs research, indicators and associated thresholds have been established in premenopausal/andropausal able-bodied adults in Westernised countries and laboratories, and, thus, potentially lack full validation in various global racial and ethnic populations, or para sport and transgender athletes. Nevertheless, where appropriate, the elements and approach of this tool can be applied to various minority populations as a scientific starting point, but appreciating the lower level of validity. It is also important to note that menstrual cycle status and endogenous sex hormone levels cannot be accurately assessed in athletes who are taking sex hormone-altering medications (eg, hormone-based contraceptives), and thyroid hormone status indicators cannot be accurately assessed in athletes who are taking thyroid medications.

## CONCLUSIONS

This IOC REDs CAT2 highlights more than 30 potential signs and symptoms (indicators) of problematic LEA leading to REDs. These indicators are scored using a tool that allows objective assessment of athletes by medical professionals. An athlete can be given a green light (healthy with full participation clearance) or a yellow, orange or red light (diagnosed with REDs with subsequent participation restrictions based on severity). Numerous LEA signs and symptoms should not be surprising, as LEA can impact every biological process dependent on energy output. Indeed, all physiological processes and associated organs/tissues related to growth (eg, low BMD, falling off the paediatric growth

curves), reproduction (eg, FHA, low sex hormones), maintenance (eg, low T3, RMR) and activity simultaneously compete for finite energy resources. Thus, inadequate EI or an increased energy commitment to one metabolic process creates energetic 'trade-offs' away from other processes.[149 180]

In conclusion, the pursuit of identifying better differentiating LEA indicators needs continued investigation, appreciating there will be different indicators for females vs males, and para vs able-bodied athletes, across varying timelines, at different biological and gynaecological ages, with varying LEA levels, and with consideration of many other moderating factors. Further severity/risk assessment stratification and diagnostic advancements will result from further understanding of the complex multifactorial mosaic of LEA signs and symptoms.

**Correction notice** This article has been corrected since it published. Figure 2 has been updated in the online version only and not in print.

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# Correction: *Review of the scientific rationale, development and validation of the International Olympic Committee Relative Energy Deficiency in Sport Clinical Assessment Tool: V.2 (IOC REDs CAT2)—by a subgroup of the IOC consensus on REDs*

Stellingwerff T, Mountjoy M, McCluskey WT, *et al*. Review of the scientific rationale, development and validation of the International Olympic Committee Relative Energy Deficiency in Sport Clinical Assessment Tool: V.2 (IOC REDs CAT2)—by a subgroup of the IOC consensus on REDs. *Br J Sports Med* 2023;57:1109-18. doi: 10.1136/bjsports-2023-106914
Figure 2 and online supplemental appendix 1 have been updated. This has been updated in the online version only and not in print.

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